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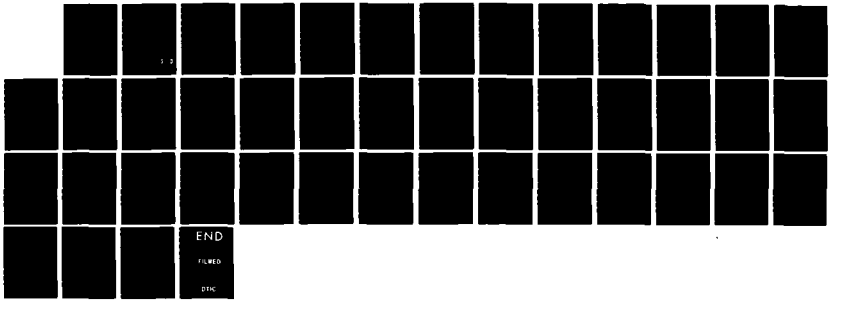

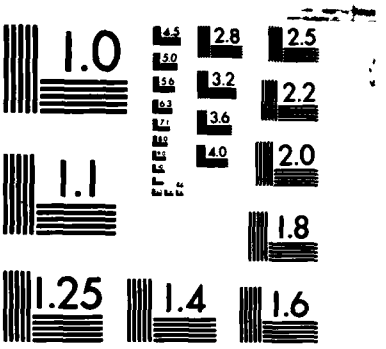

PHYSIOLOGICAL EFFECTS OF TRAINING

by

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With the evolution of exercise science a vast amount of information concerning the physiological effects of training has been generated. Understanding the basic training responses and adaptations of various modes of conditioning can give the clinician insights into exercise prescription. The purpose of this manuscript is not to present an exhaustive review, but provide the reader with a basic overview of the physiological effects of training.

AEROBIC TRAINING

Aerobic training results in a number of adaptations in humans. The magnitude of this response is dependent upon a number of factors. It depends upon the type, the intensity, the frequency and the duration of the training; as well as the characteristics of the person undergoing training.

In this section, we will discuss the physiological changes associated with the adaptation to training and how the characteristics of the individual and the training itself affect this adaptation.

In order to determine the response to training, a measure of aerobic fitness is required. The best and most commonly measured laboratory assessment of aerobic fitness is the maximal oxygen uptake ($\dot{V}O_2max$). Astrand[1] defines $\dot{V}O_2max$ as "a measure of (1) the maximal energy output by aerobic processes, and (2) the functional capacity of the circulation..." $\dot{V}O_2max$ is used to assess levels of aerobic fitness and the effects of training on aerobic fitness because it is a reliable and reproducible measure. However, in order to measure $\dot{V}O_2max$ a fair amount of expensive laboratory equipment is required. O_2 consumption can be measured while performing almost any exercise, however, for laboratory purposes the exercise is usually performed on a motor driven treadmill or a cycle ergometer. Figure 1 illustrates a standard treadmill procedure for measuring $\dot{V}O_2max$. The values shown (approx

45 ml/kg.min) represents a normal value for the average male 20-30 years of age. Oxygen consumption is usually reported as an absolute value or relative to body weight. Absolute refers simply to the amount of oxygen used each minute, for example 3.0 L/min, whereas for activities which require an individual to move his body weight (i.e., walking, running), values are best reported relative to body weight. In this case, someone who is walking, using 3.0 L of O_2/minute, and has a body weight of 60 kg would be using 50 ml/kg.min of oxygen to perform this activity. This is derived in the following manner:

$$\frac{3.0\ \text{L/min}}{60\text{kg}} = \frac{3{,}000\text{ml/min}}{60\text{kg}} = 50\ \text{ml/kg.min.}$$

Athletes generally have higher values for $\dot{V}O_2$max than their sedentary counterparts.[2,3]. Also an athlete in an endurance type sport will generally have a higher $\dot{V}O_2$max than other athletes. [1,2]

Numerous studies have reported increases in $\dot{V}O_2$max as a result of aerobic training [4,5,6,7]. This response to training is affected by several factors. The individuals' initial level of fitness is one of these factors. The effect that initial level of fitness has on the response to training was classically demonstrated by Saltin et al.[8] They reported that 5 subjects showed a 27 percent decrease in $\dot{V}O_2$max as a result of 20 days of bed rest. Subjects then went on a 50 day training program. Three of these subjects had been previously sedentary and two were physically active prior to the study. The three sedentary subjects increased their $\dot{V}O_2$ max by 100% over the post bed rest levels. The previously active subjects increased $\dot{V}O_2$max by 34% above post bed rest levels. When compared to initial levels of fitness, however, the sedentary and active subjects increased by 33% and 4%, respectively, as a result of the 50 day training program.

In another study, Daniels et al[9] reported increases, no change and decreases in the $\dot{V}O_2max$ values of West Point cadets during a six-week basic training program. The response to training was dependent upon the initial level of aerobic fitness, with those having the lowest initial level of aerobic fitness showing an increase in $\dot{V}O_2max$ and those with the highest initial level of fitness showing a decrease in $\dot{V}O_2max$ as a result of training. This decrease appeared to be due mainly to an increase in lean body weight. It should be pointed out, however, that all three groups improved their performance in the 1.5 mile run as a result of training.

Thus, the initial level of fitness of the individual is an important factor to consider when evaluating the response to aerobic training. Generally, the lower the initial level of fitness the greater the percent increase in $\dot{V}O_2max$ as a result of training. The individual's genetic endowment may also affect this initial level of fitness and the response to training.[1]

The intensity, frequency and duration of exercise are other factors that affect the response to training. The American College of Sports Medicine recommends a frequency of 3-5 days per week at an intensity between 50-85% of $\dot{V}O_2max$ for a duration of 15-60 minutes in healthy adults.[7]

Improvement in $\dot{V}O_2max$ in the range of 5% to 25% can be expected as a result of a moderate training program.[6,9,10] Improvements greater than this have been reported but these are associated with low levels of initial fitness[8] or very intense and strenuous training programs.[11] The levels chosen by the American College of Sport Medicine represent those levels for intensity, duration and frequency that will provide the non-athlete with an adequate training stimulus without unnecessary risk of injury.

Studies have shown that increasing the intensity of exercise raises the drop out rate in the non-athlete runner.[3] However, the importance of adequate intensity was shown by Karvonen et al [12]. They showed that in two groups of young men training on a treadmill, there was no improvement in the group that ran at a heart rate of less than 135 beats/minute; whereas significant improvement occurred in the group training above 153 beats/minutes.

Milesis et al[13] illustrated the effect of increased duration (15-30-and 45-minutes) of training on aerobic fitness. They found significantly greater improvement in $\dot{V}O_2max$ in the longest duration group (45 minutes) compared to the 15-minute group.

Similar results were reported by Wilmore et al[10] when comparing 12 and 24 minutes of exercise, 3 days per week for 10 weeks. While both groups showed significant increases in $\dot{V}O_2max$, the increase was greater in the 24 minute group.

The increase in $\dot{V}O_2max$ with training also has been shown to be affected by the frequency of the training. In a study which compared two days a week and four days a week of training, Pollock et al[14] found twice as much of an increase in $\dot{V}O_2max$ (35% vs 17%) in the four day a week group. In another report, Pollock[3] also reported increased improvement in $\dot{V}O_2max$ as training increased from 1 to 3 to 5 days per week. However, in this same report he also noted a marked increase in the incidence of injury as the frequency increased beyond 3 days per week and when the duration per day increased above 30 minutes.

To summarize thus far, heredity and the initial level of fitness of the individual, the intensity, duration and frequency of the training program affect the response of the individual to a training program. Because of the

increased risk of injury, individuals who are just starting out are advised to begin at the lower intensity, duration and frequency levels given in the guidelines above. It must also be remembered in the discussion to follow that these factors may modify the changes in various organ systems.

Two other factors, age and sex, should also be mentioned when discussing physiological changes associated with aerobic training. $\dot{V}O_2max$ decreases with age in adults. This has been demonstrated by Astrand[15] and more recently by Vogel et al[16]. Vogel et al reported a decrease in $\dot{V}O_2max$ at an average yearly rate of 0.5ml/kg.min. in various groups of U.S. military personnel. This agrees with the average yearly decline found by Astrand et al[17] in thirty-five females and thirty-one males studied in 1949 and again in 1970. Their average yearly decline was .438 ml/kg.min and .638 ml/kg.min for females and males, respectively.

Females, on average, have $\dot{V}O_2max$ values that range from 73-85% of the values of males.[9,16,17,18,19] The physiological response to training is similar in both sexes. The most noted difference is that females often show a larger increase in $\dot{V}O_2max$ as a result of training than their male counterparts.[9,18,19] However, this is often due to the relatively lower initial level of fitness. The difference in $\dot{V}O_2max$ between the sexes has been related to several factors, all of which probably have some bearing. These include higher percent body fat in women, lower blood hemoglobin in women and larger heart size and blood volumes in men.[1,16,20]

$\dot{V}O_2max$, therefore, is the laboratory measure most commonly used to assess aerobic fitness and the effect of an aerobic training program. In the remainder of this section, we will discuss the physiological changes that occur as a result of training and which are associated with the increased

aerobic fitness. We will center our discussion around the cardiovascular, cellular and metabolic changes that occur in conjunction with an increase in $\dot{V}O_2max$.

For an in depth discussion of the cardiovascular changes associated with exercise and physical training, the reader is referred to the reviews by Bevegard and Shepherd,[21] Rowell,[22] Clausen,[23] and Scheuer and Tipton.[24] $\dot{V}O_2max$ is equivalent to the product of maximal cardiac output and arterial-venous oxygen difference or:

$\dot{V}O_2max$ = maximal cardiac output x A-V O_2 difference.

Since cardiac output is equal to heart rate times stroke volume, oxygen consumption at any level of exercise can be calculated as follows:

$\dot{V}O_2$ (oxygen consumption) = (heart rate x stroke volume) x A-V O_2 difference.

Aerobic training could result in a change in $\dot{V}O_2max$ by altering any one of these variables. In reality, changes occur in all three. Heart rate is generally reduced at all submaximal exercise levels including rest as a result of training. Wilmore[2] states that..."resting heart rate will be reduced by 1 beat/minute for each week of participation in a moderate exercise program for previously sedentary individuals." Although this statement applies only to short-term programs, the resting heart rate is normally reduced as a result of aerobic training in all age groups.[10] Studies with rats indicate that after training the decreased heart rate may be due to increased parasympathetic

activity.[24] In addition to lower resting heart rates, training results in lower heart rates at similar exercise loads.[23,24] For example, Pollock et al[25] reported a decrease of 10 to 21 beats per minute in the heart rate of middle aged men on a standard treadmill run (6.0mph, 2.5%grade) as a result of 20 weeks of running. Yoshida et al[26] reported similar findings after 8 weeks of training on a cycle ergometer. Maximal heart rate is either unchanged or slightly reduced as a result of aerobic training. This result is common in both sexes and across age groups.[9,10,11,19,26,27] While the decrease in heart rate at rest is due to increased parasympathetic drive, the decrease during submaximal and maximal exercise is believed to be due to decreased sympathetic drive.[23,28] Cardiac output does not change substantially from the pre to post-training state at the same absolute workload.[1,20,28] Therefore in order to maintain cardiac output in conjunction with a decreased heart rate, stroke volume has to increase. Stroke volume increases in the transition from rest to exercise in all healthy subjects up to a certain heart rate. Beyond a certain heart rate (approx. 110 beats/min)[28] the stroke volume falls because the end-diastolic volume becomes smaller. This is due to the decreased filling time available. Trained individuals have higher stroke volumes at rest, submaximal and maximal exercise.[1,26] Therefore, the same cardiac output is maintained at a reduced heart rate making the heart more efficient. At maximal workloads, cardiac output after training is increased significantly because stroke volume is increased and heart rate is either unchanged or only slightly decreased compared to untrained. Rowell[22] reported that increased stroke volume accounted for approximately half of the 15% increase in $\dot{V}O_2max$ that he saw in sedentary subjects after 3 months of training. Keul et al[28] recently described how moderate and intensive endurance training affect stroke

volume. Basically, they found that moderate training resulted in a reduction in adrenaline and noradrenaline (sympathetic drive) at the same absolute work load. This resulted in a decreased heart rate and arterial pressure. This allowed for more intensive filling of the heart and a larger end diastolic volume, as well as, a decreased afterload on the heart which allowed for a smaller end systolic volume. The overall effect is an increased stroke volume without any change in heart size. More intensive training results in an increase in heart size. Again the increase in stroke volume is partially due too increased end diastolic volume. Thus, the pumping ability of the heart, i.e. increased stroke volume, is improved with training; but there is only presumptive evidence of increased contractility of cardiac muscle.[24]

As indicated above, Rowell[22] reported that approximately half of the increase in $\dot{V}O_2max$ could be accounted for by increased stroke volume. The remainder of the increase was due to an increase in the A-V O_2 difference. Adams et al [29] report similar results. This increase could be due to two factors. First of all, the cardiovascular system may have become more efficient in supplying the exercising tissue with O_2 or secondly, the tissue may be better adapted to the use of O_2 with which it is provided.

Brooks[20] reports that after training blood flow to active muscle is either unchanged or slightly reduced. Clausen[23] also presents evidence that during submaximal exercise, blood flow to active muscle is reduced. This, as well as other experimental findings to be discussed, strongly support the contention that trained muscles extract more oxygen from the blood. However, during maximal exercise, the blood flow to exercising muscle is also increased in the trained state.[20,23]

Scheuer and Tipton[24] suggested several possible mechanisms that might be responsible for the increased A-V O_2 difference with training. These included changes in "...muscle blood flow, number of capillaries, the fiber type of muscles being recruited, alterations in concentration and activity of aerobic enzymes in cells, plus changes in the number and function of the mitochondria."

Research, to date, documents a number of changes in muscle cells as a result of training. These biochemical changes increase the ability of cells to perform aerobic metabolism. Thus, while there is no direct evidence to conclusively link these cellular adaptations to the increase in A-V O_2 difference, data strongly suggests that this is the case.

The remainder of this section will be devoted to the cellular and metabolic changes associated with aerobic training. Reviews by Holloszy,[30] Holloszy and Booth[31] and Howald[32] have discussed the biochemical, morphological and functional changes associated with aerobic training. Aerobic training results in a number of changes in muscle tissue itself. These changes include increases in the density of capillaries supplying muscle fibers, increases in mitochondrial densities, changes in the substrates utilized for aerobic metabolism, increases in enzyme activities supporting oxidative reactions and an increase in myoglobin content of muscle. Some studies also indicate that training can result in changes in muscle fiber type distribution patterns.[33,34] All of these changes assist in the maintainence of cellular homeostasis during prolonged exerecise.

The number of capillaries per unit of fiber has been reported to increase with physical training and to be higher in endurance trained athletes.[35,36] Appell[37] presents evidence to indicate that the number of capillaries does not

change but that adaptations occur in capillaries as a result of training that cause them to travel a more "tortuous" route and this augments their cross-sectional area.

The increase in the mitochondrial densities as a result of aerobic training increases the oxidative capacity of trained muscle. Davies et al[38] reported a 100% increase in the mitochondrial content of muscle and in the tissue oxidative capacity as a result of 10 weeks of training in rats. They reported a strong relationship between the muscle oxidative capacity and endurance performance. Holloszy and Booth[31] summarized the mitochondrial enzyme level increases that occur as a result of endurance training. These enzymes are involved in the activation and oxidation of fatty acids, in the Krebs cycle and in the electron-transport chain. Holloszy[30] listed the increases in the specific enzymes that occur as a result of aerobic training. Training increased the levels of activity of enzymes (palmityl CoA synthetase, carnitine palmityl transferase and palmityl CoA dehydrogenase) involved in the activation, transport and catabolism of long chain fatty acids. Citrate synthase, DPN-specific isocitrate dehydrogenase and succinate dehydrogenase; all enzymes involved in the Krebs cycle, showed a twofold increase in their level of activity as a result of endurance training. Davies et al[38] reported a 108% increase in the mitochondrial content of succinate dehydrogenase.

Increases in the activity of components of the electron transport chain have also been shown to occur with endurance training. Several authors[20,30,31,38] have indicated that as a result of endurance training, the number and the content of muscle mitonchondria are increased. The increase in activity levels, however, is an increase in total muscle activity, rather than an increase in the specific activity of the individual enzymes. Brocks and

Fahey[20] present data which shows that while mitochondrial content is increased, the specific activity of the individual components of the electron transport chain is unaffected by endurance training.

According to Holloszy[30] "...when skeletal muscle adapts to endurance exercise, it becomes more like cardiac muscle in that its content of mitochondria and its capacity to generate ATP from oxidation of pyruvate and fatty acids increases."

The net result of this incresae in the oxidative capacity of skeletal muscle is that submaximal exercise in the trained state causes less of an alteration in cell homeostasis. The increased capacity for fatty acid oxidation results in a slower utilization of glycogen by working muscles in the trained state. This helps prevent the depletion of glycogen which has been demonstrated to be one of the factors limiting prolonged exercise.[39,40] The glycogen sparing effect associated with training has been demonstrated by Saltin and Karlsson.[41] They found that 6-8 weeks of endurance training resulted in a slower depletion of muscle glycogen for the same workload in five subjects. They also found that at the same relative per cent of $\dot{V}O_2$ max, well-trained subjects were using glycogen at a similar rate despite the fact that they were working at a higher absolute oxygen uptake. This was associated with a lower blood lactate level. One of the metabolic effects of aerobic training is a change in the workload, both absolute and per cent of $\dot{V}O_2$ max, at which lactate begins to accumulate in the peripheral blood.[42] Gleser and Vogel[43] found that endurance time varied inversely with level of blood lactate. Blood lactate levels have been shown to be a useful measure for predicting the ability of individuals in endurance performance.

Costill et al[44] and Costill[45] noted that trained distance runners were capable of running at speeds much closer to maximum than untrained individuals before showing elevations in blood lactate. Since then several investigators[46,47] have shown high correlations between some measure of blood lactate accumulation and running performance. Other investigators [48,49] have used the concept of "anaerobic threshold" and found strong correlations with running performance. The concept of "anaerobic threshold" was introduced by Wasserman and McIlroy.[50] According to their theory, at low exercise intensities , lactate is not produced by exercising muscle and blood lactate levels are the same as at rest. At some exercise intensity, blood lactate concentration begins to increase and as exercise intensifies, blood lactate progressively rises until the maximum effort is achieved. Furthermore, the theory states that these changes in blood lactate can be determined by changes in ventilatory parameters. This is based on the assumption that the rise in blood lactate concentration results in an exponential increase in ventilation as the ventilatory control mechanisms attempt to buffer lactic acid by blowing off "excess" CO_2. Lactic acid formed in muscle almost completely dissociates to hydrogen ion(H^+) and lactate. The H^+ produces CO_2 thru the following reaction:

$$H^+ + HCO^- = H_2CO_3 = CO_2 + H_2O$$

(bicarbonate) (carbonic acid) (carbon dioxide).

They felt that the increase in blood lactate was linked to the onset of local muscle hypoxia at some workload. The word "anaerobic" was used to indicate that the supply of O_2 was not sufficient to meet all of the muscle energy

demands by aerobic metabolism. Recently, considerable controversy has occurred over the theory that the muscle is hypoxic and also whether ventilatory parameters are a valid indicator of lactate accumulation. The concept of anaerobic threshold was recently reviewed by a supporter[51] and an opponent[52] of the theory. Brooks[52] does not think that the accumulation of lactate is caused by muscle tissue becoming anaerobic. He presents evidence to demonstrate that there is sufficient O_2 present to carry on aerobic metabolism. He believes lactate accumulation is not due to a sudden increase in lactate production but rather it"...is the result of (1) those processes which produce lactate and contribute to its appearance in the blood and (2) those processes which catabolize lactate after its removal from the blood." Brooks reports that, in the rat, the major effect of training on lactate metabolism is an increase in lactate clearance. This has not been demonstrated in humans.

Regardless of the outcome of this controversy, lactate accumulation in blood has become a useful measure for predicting endurance performance. However, the cause and the mechanisms responsible for the accumulation and how these mechanisms are affected by training remains to be defined.

Other metabolic effects of training center around the enhancement of mobilization and utilization of free fatty acid and the glycogen sparing effect discussed earlier. Since an in depth discussion of these change is beyond the scope of this paper, readers are referred to the following reviews.[53-55] Basically, catecholamines, insulin and glucagon are involved in regulating lipolysis, hepatic glycogenolysis and gluconeogenesis. Galbo[53] reports that in trained and untrained subjects the hormonal response depends on the relative rather than the absolute workload.

Other factors which can be favorably affected by aerobic training but which will not be discussed here include body composition,[56] cardiovascular risk factors[57] and psychological well-being.[58]

In summary, aerobic training results in a number of adaptations which improve not only maximal performance but also the ability to do prolonged exercise. The amount of improvement depends upon the characteristics of the individual and the training that is undergone. One of the most commonly measured changes is an increse in the maximal oxygen uptake. This is associated with an increase in the maximum cardiac output. Generally, at submaximal workloads, heart rate is decreased and stroke volume increased when compared to the untrained state. Metabolic changes include increased respiratory capacity in skeletal muscle, increased mitochondrial density and enzyme activity and lower blood lactate levels at a given workload. There is also a shift towards the use of fatty acids for fuel which results in a glycogen sparing effect in muscle. As a result of these adaptations, the cardiovascular system is generally considered to be more efficient and exercise causes less of a disturbance in the homeostasis of skeletal muscle.

RESISTANCE TRAINING

Resistance training is a popular form of conditioning in rehabilitation, fitness and athletics. The term "strength training" has been coined from the obvious objective of most resistance exercise programs. For the purpose of this manuscript the term "strength" will refer to the muscle's ability to produce force.[59]

The physiological alterations consequent to resistnace training are not just strength improvement. Futhermore, all resistance training programs can not be classified as being the same. Depending on the choices made for

program variables such as the choice of exercise, order of exercise, number of sets, load used and duration of rest allowed, resistance training programs can be quite diverse.[127,130] The extent of physiological variation in response to changes in these program variables remains speculative but probably runs along a continuum.[65] The vast number of programs possible with simple manipulations probably helps explain why there are so many possible resistance training programs.

Due to variations in pre-training status of subjects, program variables and the duration of the training period, it has been difficult to make more than general observations on the apparrent effects of specific resistance training programs. In the attempt to examine the physiological effects of chronic resistance training programs, cross-sectional studies of competitive lifters (olympic lifters, powerlifters and bodybuilders) have been undertaken. Certain limitations are inherent with this type of research including subjective groupings of various caliber of lifters for statistical analysis, lack of a universal definition for "elite" lifters by researchers and possible augmentation of exercise stimulus with the use of anabolic steroids. Still it appears to be our best model to examine long term training effects.

The classic response which has been observed in most studies examining resistance training is the increase in muscular strength.[63,64,65,126,129,144] Most of the research over the past four decades had focused on the program variables of the number of sets, and the load used and its relationship to strength gains.

It is pretty well established that multiple sets of an exercise is superior to single set systems for development of maximal strength.[62,-66] Most research indicates the use of 3 to 6 sets of a particular load.[65] It

would appear that deminishing returns occurs after 6 to 8 sets.[135,136,137] Yet it is improtant to remember that these observations are for relatively novice subjects who may not have been able to tolerate the greater volume (sets x repetitions x load) of exercise stress. For experienced lifters variation in volume of training may be essential for optimal gains in strength.[131,134]

Data from research concerning the optimal load for maximal strength gains generally suggests that gains occur with the use of a 5 or 6 repetition maximum (RM) loading.[65,135-142] Again this observation is inferred from subject populations who are essentially novice weight trainers after a short term program. A load continuum exits.[63,65,127,124] Generally, heavy loads (1RM to 6RM) results in the greatest strengths gains. Use of moderate (7RM to 12RM) to light (greater than 12RM) produces strength gains but the magnitude of the increase is as one moves to the lighter loads on the continuum. Data from Anderson and Kearney[70] supported this concept. In their study it was demonstrated that greatest strength gains occur with the heaviest loads and deminished with lighter loads. Atha[65] suggests that minimal returns on strength occur at about 20 to 25 RM loads.

Training is typically performed with 24 to 48 hours rest between workouts.[162,163] The major concern is that adaquate recovery is allowed between workouts. In athletes with a great deal of training background it is not unusual for workouts to be performed six days a week. A recent study by Hunter[89] demonstrated that four straight days of training in a row to be superior to alternate day workouts. This may be related to the recovery phenomenon and does require further study.

Hakkinen[68] has demonstrated the importance of pre-training status as a determinant of the magnitude of strength increase. It was shown that nonathletes accustomed to resistance training made increases in strength to nearly twice the degree of weight trained athletes in half the time. This again suggests that with an individual of low functional capacity, gains are made easily and as one gets closer to their genetic potential, more work is needed to make even small improvements.

As training periods extend to longer periods of time (i.e. over a year) the type of workouts used may need to be varied.[134] This concept has been called "cycling" or "periodization" of training.[131] By changing the workout intensity (load) and volume (sets x repetitions x load) over the course of a training period, the muscle(s) apparently responds with greater maximal strength improvement. When compared to conventional resistance training programs, periodization of training even in a short term program appears to be superior for development of maximal strength.[128]

The various types of strength training contraction styles (i.e. isometric, dynamic concentric/eccentric, isokinetic) have been extensively reviewed by Fleck and Schutt.[64] Each contraction style may be appropriate for different situational demands. For example, isometric programs may be appropriate in rehabilitation where pain or injury limit the range of joint motion. Dynamic programs may be more appropriate for athletes who need to train the entire joint motion maximally.[147,148] For more definitive reviews on contraction styles and terminnology the reader is referred to the following references [59,63,65]. The effects of different contraction styles of training is related to the inherent differences between them. One must be aware of the advantages and disadvantages of each style of training. Clinical decisions

need to be made for each individual case based on the need, safety, range of motion of the strength gains and effectiveness of the exercise stimulus.

Thus, development of an optimal program for improvement of strength is dependent on the initial fitness level of the subject,[87] high intensity overload of the muscle[63] and variation in the exercise stimulus to maintain effectiveness.[131]

Cellular Changes

Strength training causes an increase in the growth of muscle.[91,102,103] This process called hypertrophy has typically been attributed to increaes in the cross-sectional area of muscle fibers, the increase in the size and number of actin and myosin filaments and addition of sarcomeres to existing muscle fibers.[69,85,98,104,146] A lot of controversy has surrounded the concept of hyperplasia (the increase in the number of fibers) which could account for the increased size of muscle observed following different strength training programs. Initial evidence for hyperplasia was presented in animal studies.[84,88] Over the past five years examination of bodybuilders revealed that the cross-sectional size of muscle fibers in the triceps brachii were not any different than untrained controls.[99,118] It appeared that since the arms of these athletes were significantly larger than controls, the adaptation may be the result of an increase in the number of fibers. A recent study of the biceps brachii of bodybuilders contradicted previous evidence in the triceps brachii and thus the issue remains equivocal.[176]

Different strength training workouts seem to result in a significantly different response when examining the capillary density of muscle. Powerlifters and olympic style lifters tipically perform workouts which are high intensity and allow for longer rest periods (2 to 5 min) betweeen sets

contrasted with bodybuilders who use moderate to high intensity loads, allow very little rest between sets and probably perform more exercises. The data from the bodybuilders suggests that this type of training program may induce capillary growth whereas powerlifting and olympic lifting styles of training may reduce it.[115,119] The physiological reason for this adaptation and the exact stimulus requires more definitive study. How this may ralate to aerobic function or cardiovascular fitness remains speculative.

Strength training appears to decrease the volume density of mitochondira. In a study by MacDougall et al.[100] steriological analysis indicated that the mitochondrial volume density in the triceps brachii decreased following a six month high intensity resistance training program. The 26% reduction in mitochondrial volume density seemed to indicate that heavy resistance training programs may not be suitable for those individuals concerned with aerobic performance. Yet the limited data on compatibility of aerobic metabolism and resistance training does not seem to support this fear as no decreases in aerobic capacity have been reported.[20] More study is needed to further understand the magnitude of this response in other muscles and when using other resistance programs with lower intensity.

Nervous System

The effects of resistance training on the nervous system response is an important factor becuase of the functional relationship between the nerve and the muscle.[120] Using an integrated electromyogram (EMG) methodologies, Moritani and DeVries demonstrated that significant strength gains can take place in the absence of any significant tissue hypertrophy in the initial stages (2 to 4 weeks) of a training program.[108] The neural component appears to be the primary mechanism underlying initial strength gains. Yet the

hypertrophy component still made the largest contribution to the increased strength observed in young adult males following training.[108] This was constasted with older males whose increased strength was explained by a greater involvement of the nerual component throughout the training period.[109] It would appear that strength gains are mediated by a different neural response to training as one grows older. It has been proposed that females may utilize similar nerual mechanisms to achieve strength gains.[20] The effects of resistance training in females is not significantly different from males except for initial upper body strength levels and obvious hormonal differences which may be responsible for the lack of excessive hypertrophy observed in some males.[60]

Data suggests that the nervous system plays a very large role in the possible mediation of strength gains.[77,92] This may include an improved recruitment pattern, increased synchronization, longer tonic activity and less inhibition.[73,93,106,113] The efficacy of these mechanisms in different muscles and the responses to different styles of resistance training remains unknown. Still it is clear that resistance training significantly alters nerual function.

Bioenergetics and Enzyme Activities

Limited data suggests that strength training increases enzyme activities involved with splitting of energy rich phosphates, anaerobic glycolysis and glycogenolysis along with oxidation of carbohydrates.[1,20,32] This is associated with increases that have been demonstrated for high energy phosphates and glycogen following training. The main energy source for high intensity resistance training exercise is primarily anaerobic.[1] Thus, it would appear that most adaptive changes are associated with the ability to

support anaerobic functions.[76,86,114] This is an attractive hypothesis but one should be aware that it has been demonstrated that aerobic enzyme markers can increase following anaerobic training. Howald[32] maintains that "the metabolic adaptations occurring in the different fiber populations are strongly related to their recruitment pattern during exercise." With recent advances in single fiber analysis of enzyme activities, it has been shown that most vary over more than a ten fold range implicating a spectrum of metabolic profiles with significant overlap between fibers.[32] Thus, contractile properties do not always determine metabolic capacities. A great deal of study is still needed to determine the responses to different resistance training programs.

Cardiovascular Fitness

The ability to increase aerobic fitness using a resistance training program has been of interest to both scientist and practitioner.[117] Circuit weight training programs utilizing light loads and low rest periods between sets and exercises were the primary programs used to examine this possibility.[71,90,123] Gettman and Pollock[67] examining the various studies concerning aerobic changes with circuit weight training found only moderate increases (5 to 8%) in maximal oxygen consumption to occur following this style of training. A recent study by Hurly et al.[90] demonstrated no significant improvement in aerobic capacity was realized when a high intensity single set circuit was used. The potential lack of aerobic stimulus for this type of express circuit training was further supported in a subsequent study examining the heart rate and oxygen consumption demands.[158] The responses were well below recommended minimal aerobic training intensity levels. Only moderate gains are realized in aerobic function using resistance training and

are highly dependent on the fitness level of the subject.[81,82,83] It is unlikely that endurance trained individuals would enhance aerobic fitness by using resistance training.[166]

Preventive medicine as it relates to diet and weight control habits along with exercise have gained quite a lot of attention over the past ten years and have been linked to cardiovascular health. Most studies have demonstrated that resistance training can make effective changes in the body composition of an individual. The typical response is an increase in lean body mass and a reduction of the fat content.[20,61] The use of more strenuous workout schedules may even produce more drastic changes as evidenced by the low body fat percentages (8 and 13%) observed in male and female competitive bodybuilders.[80,95,111,116,125]

Elevation of certain blood lipids have been linked to cardiovascular disease. Aerobic exercise appears to reduce those blood lipid levels and thus reduce potential cardiovascular risk.[56] Conversely cross-sectional studies of competitive lifters have failed to demonstrate a similar response for chronic resistance training exercise as blood lipid levels were no different than nonathletes.[78] This seemed to implicate a less effective role for modifying blood lipids. Hurly, et al.[159,168] demonstrated that training programs used by body builders is associated with a more favorable lipid profile than the training programs used by powerlifers. This might be related to the potential higher metabolic cost of bodybuilding workouts compared to power lifting workouts. In addition, it was shown that the use of anabolic steroids may increase the risk of coronary disease do to the adverse effects on lipid profiles. Goldburg, et al.[160] did recently demonstrate that weight training could have a favorable result on the blood lipid levels of previously

sedentary men and women (mean age 33 and 27 respectively). Significant reductions were observed in the absolute values of low-density lipoprotein (LDL) cholesterol and triglycerides, along with the ratios of total cholestoral-high-density lipoprotein (HDL) cholesterol and LDL cholesterol-HDL cholesterol. Thus, resistance training may be effective in reducing blood lipid levels, but not with concurrent use of anabolic steroids.

Reduction of resting blood pressure is a common response to aerobic training presumably associated with improvements in cardiovascular fitness and health.[28,57] Converesly, resistance training has never been viewed as positively affecting resting blood pressure due to the large pressor response typicially elicited with lifting activities. Furthermore much less is known about resting and exercise blood pressure responses to resistance exercise. MacDougall, et al.[101] has reported the extreme elevations in arterial blood pressure response (255/190 to 320/250) associated with the performance of a single arm curl and a double leg press. In a recent resport by Fleck and Dean[79] extreme increases in resistance exercise arterial blood pressure were observed but not to the same magnitude as the previous study. This dispariety may be due to the elimination of the Valsalva maneuver usually associated with weight lifting exercise. Furthermore the level of the competitive bodybuilder used in each study may have been different.

The relationship of the exercise response to resting blood pressure has not been fully examined. Resting blood pressure responses of competitive lifters are still not reported as being abnormal or hypertensive.[79,97,101] Short term resistance training programs have not resulted in increases in resting blood pressure response. Yet, in a report by Hunter and McCarthy[152] it was demonstrated that not all individuals can tolerate intense anaerobic

training. Resting systolic blood pressures significantly increased over the course of an intense cycling and resistance training program. This response may have been related to an overtraining syndrome and lack of any periodization of training. The responses of individual subjects were variable and again pointed to the need for individual monitoring and prescription.

Hormonal Response

At the present time very little is known about the hormonal response to different resistance training programs. Most of the data has been generated from partial workouts or a single exercise response. The physiological role of hormones is typically connected with metabolic responses and growth.[53,54,172]

It has been suggested that testosterone may be related to both strength and indirectly involved in the augmentation of exercise induced-hypertrophy. Few studies have examined the responses of testosterone in men and women to resistance training exercise. Weiss et al.[175] described testosterone responses in men and women following a four station exercise circuit (latissimus pull down, supine bench press, arm curl and leg press) with a 2 minute rest period between sets and exercises. Subjects exercised at 80% of their 1RM. Men significantly increased testosterone levels following the circuit and women did not. It was concluded that men have greater absolute testosterone responses to resistance training. These results may have implications for differences observed in muscle with hypertrophy associated with chronic resistance training in men and women. Other studies have demonstrated similiar findings but have been unable to link resting testoserone levels to strength.[167,171] Skierska[171] did not report any increases in testosterone following a 30 minute weight lifting workout in

highly trained lifters but this study failed to report intensity levels and if subjects were using anabolic steroids. These data also suggest that intensity used plays an important role in testosterone's response to lifting exercise.

Growth hormone appears to be sensitive to intensity and frequency of lifting.[169,173] It is involved with both the metabolic and growth process.[170] Data from Vanhelder et al.[173] suggests that when using a single leg press exercise load and frequency of exercise are determining factors involved with the response of growth hormone levels. It is noted that muscle lactate and oxygen deficit may also play regulatory roles in the response of growth hormone. A considerable amount of research needs to be done to gain greater understanding of the interface between the hormomal responses and resistance training adaptations.

ANAEROBIC TRAINING - ——

In addition to resistance training, use of other conditioning programs such as sprint running or cycling are also anaerobic in nature. This type of training may have more of a direct relationship to the motor activity of a some sports. The requirement for various degrees of anaerobic endurance (moderate to high intensity) in some sports is great.[20,161] Depending upon the percent of the maximal power output used in training concurrent effects on aerobic metabolism are possible. This is evidenced by the associated cellular changes and increases in maximum oxygen consumption observed with interval sprint and cycling training which are similar to the aerobic training changes. This may bew due to the number and length of exercise intervals.[132,133] One must again remember that all effects are on a continuum so that it is difficult to determine exact thresholds for these changes. Also, the genetic predisposition of the individual plays a large role in the magnitude of the training response.[1,32]

Interval sprint or cycle training typically increases maximum oxygen consumption. Fox, et al.[151] has demonstrated that low power and high power output interval training in males elicits similar changes in maximum oxygen consumption, but that production of lactate during heavy submaximal exercise is reduced to a greater extent with a low power program. Similar responses have been observed with females for increases in maximal oxygen consumption[157] but are seemingly independent of distance and high-intensity of the interval training program.[150]

In a study by Saltin[174] it is appears that the magnitude of response for maximal oxygen consumption and aerobic enzyme activity is greater for endurance training than sprint training. The sprint leg demonstrated more FT cross-sectional diameter increases.

Interval and sprint type activities appear to significantly alter enzyme activities following training.[155] Roberts, et al.[156] demonstrated increases in enzymes (phosphofructokinase,glyceraldehyde phosphate dehydrogenase, lactate dehydrogenase, malate dehydrogenase) following sixteen training sessions consisting of 200-meter runs at 90% maxiaml speed. No increases were observed for succinate dehydrogenase. The improvement in anaerobic performance was linked to increases in key anaerobic enzyme activity. Green, et al.[165] also demonstrated that changes in enzyme profiles or fiber distributions do not occur in response to a short term stimulus period (2 consecutive days) of supramaxiaml exercise.

Changes in the energy substrates stored in the muscle is similar to resistance training and again depends on the aerobic component of the training program.[32] High energy phosphates and glycogen increase. It is unlikely that triglycerides stores are significantly increased in those programs with high anaerobic components.

REFERENCES

1. Astrand PO, Rodahl K: Texbook of Work Physiology. McGraw-Hill Bo Company, New York 1977
2. Wilmore JH: Acute and chronic physiological responses to exercise. p 53. In Amsterdam EA, Wilmore JH, DeMaria AN. (eds) Exercise in cardiovascular health and disease. Yorke Medical Books, New York, 1977
3. Pollock ML: How much exercise is enough? Physician and Sports Me 6: 50-64, 1978
4. Burke EJ: Physiological effects of similar training programs in males and females. Res Quart 48:510-517, 1977
5. Gettman LR, Pollock ML, Durstine JL, et al: Physiological responese of men to 1, 3 and 5 days per week training program. Re Quart 47:638-646, 1976
6. Pollock ML: The quantification of endurance training programs. p. 155 In Wilmore JH(ed) Exercise and Sport Science Reviews. Academic Press, New York, 1973
7. American College of Sports Medicine. Position stand on the recommended quantity and quality of exercise for developing and maintaining fitness in healthy adults. 1978
8. Saltin B, Blomquist B, Mitchell JH, et al: Responses to submaximal and maximal exercise after bed rest and training. Circulation 38(Suppl.7): 1-78, 1968
9. Daniels WL, Kowal DM, Vogel JA, et al: Physiological effects of a military training program on male and female cadets. Aviat Space Environ Med 50:562-566, 1979
10. Wilmore JH, Royce J, Girandola PH, et al: Physiological alterations resulting from a 10-week jogging program. Med Sci Sports 2:7-14, 1970
11. Hickson RC, Bomze HA, Holloszy JO: Linear increase in aerobic power induced by a strenuous program of endurance exercise. J Appl Physiol: Respirat Envrion Exercise Physiol. 42:372-376, 1977
12. Karvonen M, Kentala K, Mustala O: The effects of training heart rate: a longitudinal study. Ann Med Exptl Biol Fenn 35:307-315, 1957

13. Milesis CA, Pollock ML, Bah MD, et al: Effects of different durations of training on cardiorespiratory function, body composition and serum lipids. Res Quart 47:716-725, 1976

14. Pollock ML, Cureton TK, Greninger L: Effects of frequency of training on working capacity, cardiovascular function, and body composition of adult men. Med Sci Sports 1:70-74, 1969

15. Astrand PO: Physical performance as a function of age. J Amer Med Assoc. 205:105-109, 1968

16. Vogel JA, Patton JF, Mello RP, et al: An analysis of aerobic capacity in a large United States population. Submitted for publication.

17. Astrand I, Astrand PO, Hallback I, et al: Reduction in maximal oxygen uptake with age. J Appl Physiol 35:649-654, 1973

18. Patton JF, Daniels WL, Vogel JA: Aerobic power and body fat of men and women during Army Basic Training. Aviat Space Environ Med 51:492-496, 1980

19. Burke EJ: Physiological effects of similar training programs in males and females. Res Quart 48:510-517, 1977

20. Brooks GA, Fahey TD: Exercise physiology: Human bioeneigetics and its application. John Wiley and Sons, New York, 1984

21. Bevegard BS, Shepherd JR: Regulation of the circulation during exercise in man. Physiol Rev 47:178-213, 1967

22. Rowell LB: Human cardiovascular adjustments to exercise and thermal stress. Physiol Ref 54:75-159, 1974

23. Clausen JP: Effect of Physical Training on cardiovascular adjustments to exercise in man. Physiol Rev 57:779-815, 1977

24. Scheuer J, Tipton CM: Cardiovascular adaptations to physical training Ann Rev Physiol 39:221-51, 1977

25. Pollack ML, Broida J, Kendrick A, et al: Effects of training two days per week at different intensities on middle-aged men. Med Sci Sports 4:192-197, 1972

26. Yoshida T, Suda Y, Takeuchi N: Endurance training regimen based upon arterial blood lactate: Effects on anaerobic threshold. Eur J App Physiol 49:223-230, 1982

27. Patton JF, Vogel JA, Bodynek J et al: Response of age forty and over military personnel to an unsupervised, self-administered

aerobic training program. Aviat Space Environ Med 54:138-143, 1983

28. Keul J, Dickhuth HH, Lehmann M, et al: The athlete's heart - haemodyanamics and structure. Int J Sports Med 3:33-43, 1982

29. Adams WC, McHenry MM, Bernaouer EM: Long-term physiologic adaptations to exercise with special reference to performance and cardiorespiratory function in health and disease.p.322 In Amsterdam EA, Wilmore JH, DeMaria AN(eds) Exercise in cardiovascular health and disease. Yorke Medical Books, New York, 1977

30. Holloszy JO: Biochemical adaptations to exercise: aerobic metabolism. p.45 In Wilmore JH (ed) Exercise and Sports Sciences Review Academic Press, New York, 1973

31. Holloszoy JO, Booth FW: Biochemical adaptations to endurance exercise in muscle. Ann Rev Physiol 38:273-291, 1976

32. Howald H: Training-induced morphological and functional changes in skeletal muscle. Int J Sports Med 3:1-12,1982

33. Janssen E, Sjodin B, Tesch P: Changes in muscle fiber type destribution in man after physical training. A sign of fiber type transformation. Acta Physiol Scand, 97: 392-397, 1978

34. Tesch P, Karlsson J, Sjoin B: Muscle fiber type distribution in in trained and untrained muscles of athletes.p.79 In Komi PV (ed) Exercise and Sport Biology. Human Kinetics Publishers, Champaign, IL, 1982

35. Anderson P, Henicksson J: Capilary supply of the quadraceps femouis muscle in man: adaptive response to exercise. J Physiol 270: 677-690, 1970

36. Brodal P, Imgger F, Hermansen L: Capillary supply of skeletal muscle fibers in untrained and endurance trained men. Am J Sports Med 232: H705-H712, 1977

37. Appell HJ: Morphological studies on skeletal muscle capillaries under conditions of high altitude training. Int J Sports Med 1: 103-109,1980

38. Davies KJA, Packer L, Brooks GA: Biochemical adaptation mitochondria,muscle, and and whole-animal respiration to endurance training. Arch. Biochem Biophys 209: 539-554,1981

39. Holloszy JO, Rennie MJ, Hickson RC, et al: Physiological consequences of the biochemical adaptations to endurance exercise

in Milvy P(ed) The marathon: Physiological,medical, Epidemiological and psychological studies. Amm NY Acad Sci 301 440-450, 1977

40. Gollnick PD: Free fatty acid turnover and the availability of of substraights as a limiting factor in prolonged exercise. In Milvy P(ed) The Marathon: Physiological, medical, epidemiological and psyologicdal studies. Amm NY Acad Sci 301: 64-71, 1977

41. Saltin B, Karlsson J: Muscle glycogen utilization during work of different intensities p. 289 In Pernow B, Saltin B (ed) Muscle metabolism during exercise. Plenum Press, New York, 1971

42. Hermansen L, Saltin B: Blood lactate concentration during exercise at acute exposure to altitude. p. 48 In Margaia R (ed) Exercise at altitude. Excepta Medica Foundation, Amsterdam, 1967

43. Gleser MA, Vogel JA: Effects of acute alterations of VO2 max on endurance capacity of men. J Appl Physiol 34: 443-447, 1973

44. Costill DL, Thomson H, Roberts E: Fractional utilization of the aerobic capacity during distance running. Med Sci Sports 5: 248-252 1973

45. Costill DC: Metabolic responses during distance running. J Appl Physiol 28: 251-255, 1970

46. Farrell PA, Wilmore JH, Coyle EF,et al: Plasma lactate accumulation and distance running performance. Med Sci Sports 11:338-433, 1979

47. Sjodin B, Jacobs I: Onset of blood lactate accumulation and marathc running performance. Int J Sports Med 2: 23-26, 1981

48. Kumagai S, Tanaka K, Matsura Y, at al: Relationships of the anaerobic threshold,and onset of blood lactate accumulation. J Appl Physiol: Respirat, Environ, Exercise Physiol. 57:640-643, 1984

49. Tanaka K, Matsura Y: Marathon performance, anaerobic threshold, an onset of blood lactate accumulation. J Appl Physiol: Respirat Environ Exercise Physiol 57: 640- 643, 1984

50. Wasserman K, McILroy MB: Detecting the threshold for anaerobic metabolism in cardiac patients during exercise. Am J Cardiol 14: 844-852, 1964

51. Davis JA: Anerobic threshold: review of the concept and direction for future reseach. Med Sci Sports Exercise 17: 6-18, 1985

52. Brooks GA: Anaerobic threshold: review of the concept and directions for futher research. Med Sci Sports Exercise 17: 22-31,

1985

53. Galbo H: Endocrinology and metabolism in exercise. Int J Sports Med 2:203-211, 1983

54. Galbo H: Hormonal and metabolic adaptation to exercise. Thieme-Stratton,Inc, New York 1983

55. Winder WW: Control of hepatic glucose production during exercise. Med Sci Sports Exerc 17:2-5 1985

56. Sharkey BJ: Physiology of fitness Human Kinetics Publishers, Chicag 1979

57. Bonanno JA: Coronary risk factor modification by chronic physical exercise. p 274. In Amsterdam EA, Wilmore JH, Demaria AN (eds) Exercise in cardiovascular health and disease. Yorke Medical Books New York, 1977.

58. Folkins CH, Amsterdam EA: Control and modification of stress through chronic exercise. p280. In Amsterdam EA, Wilmore JH, DeMaria AN (eds) Exercise in cardiovascular health and disease. Yorke Medical Books, New York, 1977.

59. Knuttgen HG: Force, work, power and exercise. Med Sci Sports 10:227-228, 1978

60. Baechle TR: Women in resistance training. Clin Sports Med 3:791-807, 1984

61. Wilmore JH: Alterations in strength, body composition and anthropometric measurements consequent to a 10-week weight training program. Med Sci Sports 6:133-138, 1974

62. Berger RA: Comparison of the effect of various weight training loads on strength. Res Quart 36:141-146, 1965

63. McDonagh MJN, Davies CTM: Adaptive response of mammalian skeletal muscle to exercise with high loads. Eur J Appl Physiol, 52:139-155, 1984

64. Fleck SJ, Schutt RC: Types of strength training. Orthop Clin North Amer 14:449-458, 1983

65. Atha J: Strengthening muscle. Exercise Sport Sci Rev, 9:2-73, 198 Lesmes GR, Costill DL, Coyle EF, Fink WJ: Muscle strength and powe changes during maximal isokinetic training. Med Sci Sports 10:266-269, 1978

66. Clarke DH, Stull AG: Endurance training as a determinant of

strength and fatigability. Res Quart 41:19-26, 1970

67. Gettman LR, Pollock ML: Circuit weight training: a critical review of its physiological benefits. Phy Sports Med 9:44-60, 1981

68. Hakkinen K: Factors influencing trainability of muscular strength during short term and prolonged training. Natl Strength Cond Assoc J 7:32-37, 1985

69. Goldberg AL, Etlinger JD, Goldspink DF, Jablecki C: Mechanism of work-induced hypertrophy of skeletal muscle. Med Sci Sports 7:248-261, 1975

70. Anderson T, Kearney JT: Effects of three resistance training programs on muscular strength and absolute and relative endurance. Res Quart Exercise Sport 52:1-7, 1982

71. Allen TE, Byrd RJ, Smith DP: Hemodynamic consequences of circuit weight training. Res Quart 47:299-307, 1976

72. Bass A, Mackovg E, Vitek V: Activity of some enzymes of energy supplying metabolism in rat soleus after tenotomy of synergistic muscles and in contralateral control muscle. Phys Bohemoslovac 22:613-621, 1973

73. Belanger A, McComas AJ: Extent of motor unit activation during effort. J Appl Physiol 51:1131-1135, 1981

74. DeLorme TL, Ferris BG, Gallagher JR: Effect of progressive resistance exercise on muscle contraction time. Archs Phy Med 33:86-92, 1952

75. DeLuca CJ, Lefever RS, McCue MP, Xenakis AP: Behavior of human motor units in different muscles during linearly varying contractions. J Physiol 329:113-128, 1982

76. Edgerton VR: Mammlian muscle fiber types and their adaptability. Am Zoologist 18:113-125, 1978

77. Edgerton VR: Neuromusclar adaptation to power and endurance work. Can J Appl Sport Sci 1:49-58, 1976

78. Farrell PA, Maksucl MG, Pollock ML, et al: A comparison of plasma cholesterol, triglycerides, and high density lipoprotein-cholesterol in speed skaters, weightlifters and non-athletes, Eur J Appl Physiol 48:77-82, 1982

79. Fleck SJ, Dean LS: Influence of weight training experience on blood pressure response to exerise. Med Sci Sports Exercise 17:185, 1985

80. Freedson PS, Mihevic PM, Loucks AB, Girandola RN: Physique, body composition and psychological characteristics of competitive female body builders. Phy Sports Med 11:85-93, 1983

81. Gettman LR, Ayres JJ: Aerobic changes through 10 weeks of slow and fast speed isokinetic training. Med Sci Sports 10:[illegible]7, 1978

82. Gettman LR, Ayres JJ, Pollock MC, Durstine JL, Grantham W: Physiological effects on adult men of circuit strength training and jogging. Archs Phys Med Rehab 60:115-120, 1979

83. Gettman LR, Cutler LA, Strathman T: Physiological changes after 20 weeks of isotonic isokinetic circuit training. J Sports Med Phys Fit 20:265-274, 1980

84. Gonyea W: Role of exercise in inducing increases in skeletal muscle fiber number. J Appl Physiol: Respiration Environ Exercise 48:421-426, 1980

85. Gollnick PD, Timson BF, Moor PL, Riedy J: Muscular enlargement and number of fibers in skeletal muscles of rats. J Appl Physiol Respiration Environ Exercise Physiol, 50:936-943, 1981

86. Hakkinen K, Alen M, Komi PV: Neuromuscular, anaerobic and aerobic performance of elite power athletes. Eur J Appl Physiol 53:97-105, 1984

87. Hakkinen K, Komi PV: Changes in neuromuscular performance in voluntary and reflex contractions during strength training in man. Inter J Sports Med 4:282-288, 1983

88. Ho KW, Roy RR, Tweedle DC, et al: Skeletal muscle fiber splitting with weight-lifting exercise in rats. Am J Anatomy 157:433-440, 1980

89. Hunter GR: Changes in body composition, body build and performance associated with different weight training frequencies in males and females. Natl Strength Cond Assoc J 7:26-28, 1985

90. Hurley BF, Seals DR, Ehsani AA, et al: Effects of high-intensity strength training on cardiovascular function. Med Sci Sports Exercise 16:483-488, 1984

91. Ikai M, Fukunaga T: A study on training effect on strength per unit cross-sectional area of muscle by means of ultrasonic measurement. Eur J Appl Physiol 28:173-180, 1970

92. Ikai M, Steinhaus AH: Some factors modifying the expression of

human strength. J Appl Physiol 16:157-163, 1961

93. Kamen G, Kroll W, Zigon ST: Exercise effects upon reflex time components in weight lifters and distance runners. Med Sci Sports Exercise 13: 198-204, 1984

94. Kaneshisa H, Miyashita M: Specificity of velocity in strength training. Eur J Appl Physiol 52:104-106, 1983

95. Katch VL, Katch FI, Moffet R, Gittleson M: Muscular development and lean body weight in body builders and weight lifters. Med Sci Sports Exercise 12:340-344, 1980

96. Knakis C, Hickson RC: Left ventricular responses to a program of lower limb strength training. Chest 78:618-621, 1980

97. Longhurst JC, Kelly AR, Gonyea WJ, Mitchell JH: Echocardiographic left ventricular masses in distance runners and weight lifters. J Appl Physiol: Resp Environ Exercise Physiol 48:154-162, 1980

98. MacDougall JD, Elder GCB, Sale DG, Moroz JR, Sutton JR: Effects of strength training and immobiliation of human fibers. Eur J Appl Physiol 43:25-34, 1980

99. MacDougall JD, Sale DG, Elder GCB, Sutton JR: Muscle ultrastructural characteristics of elite power lifters and body builders. Eur J Appl Physiol 48:117-126, 1982

100. MacDougall JD, Sale DG, Moroz Jr, Elder GCB, Sutton JR, Howald H: Mitochandrial volume density in human skeletal muscle following heavy risistance training. Med Sci Sports 11:164-166, 1979

101. MacDougall JD, Tuxen D, Sale D, Moraz JR, Sutton JR: Arterial blood pressure response to heavy resistance exercise. J Appl Physiol 58:785-790,1985.

102. MacDougall JD, Ward GR, Sale DG, Sutton JR: Biochemical adaptation of human skeletal muscle to heavy resistance training and immobilization. J Appl Physiol 43:700-703, 1977

103. McDonagh MJN, Hayward CM, Davies CTM: Isometric training in human elbow flexor muscles. J Bone Joint Surgery 65:355-358, 1983

104. Martin RK, Albright JP, Clarke WR, Niffenegger JA: Load-carrying effects on the adult beagle tibia. Med Sci Sports Exercise 13:343-349, 1981

105. Menapace FJ, Hammer WJ, Ritzer TF, et al: Left ventricular size in competitive weight lifters: an echocardiographic study. Med Sci

Sports Exercise 14:72-75, 1982

106. Milner-Brown HS, Stien RB, Yemin RP: The orderly recruitment of human motor units during voluntary contractions. J Physiol 230:359-370, 1973

107. Moffroid MT, Whipple RH: Specificity of speed of exercise. Phys Therapy 50:1692-1699, 1970

108. Moritani T, DeVries HA: Neural factors versus hypertrophy in the time course of muscle strength grain. Am J Phys Med 58:115-130, 1979

109. Moritani T, DeVries HA: Potential for gross muscle hypertrophy in older men. J Gerontology 35:672-682, 1980

110. Ohtsuki T: Decrease in grip strength induced by simultaneous bilateral exertion with reference to finger strength. Ergonomics 24:37-48, 1981

111. Pipes TV: Physiological characteristics of elite body builders. Phy Sports Med 7:262-274, 1975

112. Rack DMH, Westbury DR: The effects of length and stimulus rate on isometric tension in the cat soleus muscle. J Physiol 204:443-460, 1969

113. Sale DG, McDougall JD, Upton ARM, McComas AJ: Effect of strength training upon moteneuron exitability in man. Med Sci Sports Exercise 15:57-62, 1983

114. Saltin B, Astrand PO: Maximal oxygen uptake in athletes. J Appl Physiol 23:353-358, 1967

115. Schantz B: Capillary supply in hypertrophied human skeletal muscle. Acta Physiol Scand 114:635-637, 1982

116. Sprynarova S, Parizkova J: Functional capacity and body compostion in top weight lifters, swimmers, runners and skiers. Internat Zeitschrift Fur Angewandte Physiologie 29:184-194, 1971

117. Stone MH, Wilson GD, Blessing D, Rozenek R: Cariovascular responses to short-term olympic style weight training in young men. Canadian J Appl Sport Sci 8:134-139, 1983

118. Tesch PA, Larson L: Muscle hypertrophy in body builders. Eur J Appl Physiol 49:301-306, 1982

119. Tesch PA, Thorsson A, Kaiser P: Muscle capillary supply and fiber type characteristics in weight and power lifters. J Appl Physiol:

Respiration Environ and Exercise Physiol 56:35-39

120. Thortensson A, Karlsson J, Viitasalso JHT Luhtanen P, Komi PV: Effect of strength training on EMG of human skeletal muscle. Acta Physiol Scand 98:232-236, 1976

121. Timson BF, Bowlin BK, Dudenhoeffer GA, George JB: Fiber number,area and composition of mouse soleus muscle following enlargement. J Appl Physiol: Respiration Environ Exercise Physiol 58:619-624, 1985

122. Wickiewics TL, Roy RR, Powell PL, Perrine JJ, Edgerton VR: Muscle architecture and force-velocity relationships in humans. J Appl Physiol: Respiration Environ Exercise Physiol 57:435-443, 1984

123. Wilmore JH, Parr RBG, Girandola RN, et at: Physiological alterations consequent to circuit weight training. Med Sci Sports 10:79-84,1978

124. Withers RT: Effects of varied weight training load on the strength of university freshman. Res Quart 41:110-114, 1970

125. Fleck SJ: Body composition of elite American athletes. Am J Sports Med 11:398-403, 1983

126. Delorme TL: Heavy resistance exercise. Arch Phy Med, 27:607-630, 1946.

127. Kraemer, WJ: Exercise prescription in weight training: A needs analysis. Natl Strength Cond Assoc J, 5:64-65, 1983.

128. Stowers TJ,. McMillan J, Scale D, et al: The short term effects of three different strength-power training methods. Natl Strength Cond Assoc J, 5:24-27, 1983.

129. Delorme TL: Restoration of muscle power by heavy resistance exercise. J. Bone Joint Surg, 27:665-666, 1945.

130. Kraemer WJ: Exercise prescription in weight training: manipulating program variables. Natl Strength Assoc J, 5:58-59, 1983.

131. Stone MH, O'Bryant H, Garhammer J, McMillan J, Rozenek R: A theoretical model of strength training. Natl Strength Assoc J, 4:36-39,1982.

132. Fox, EL: Sports Physiology. Saunders College Publishing, Philadelphia, 1979.

133. Fox, EL, Mathews DK: Interval Training. Saunders College Publishing, Philadelphia, 1979.

134. Matveyev L: Fundamentals of Sport Training. Progress Publishers, Moscow, 1981.

135. Berger RA: Effect of varied weight training programs on strength. Res Quart, 33: 168-181, 1962.

136. Berger RA: Optimum repetitions for the development of strength. Res Quart, 33: 334-338, 1962.

137. Berger RA: Comparative effects of three weight training programs. Res Quart, 34: 396-398, 1963.

138. Berger lRA: Comparisons of the effect of various weight training loads on strength. Res Quart, 36: 141-146, 1965.

139. Berger RA: Effect of varied sets of static training on dynamic strength. Am Corr Therapy J, 26: 52-54, 1972.

140. Berger RA, Hardage B: Effect of maximum loads for each of ten repetitions on strength improvement. Res Quart, 38: 715-718, 1967.

141. Berger RA, Harris MW: Effects of various repetitive rates in weight training on improvements in strength and endurance. J. Assoc Phys Mental Rehab, 20: 205-207, 1966.

142. Chui E: The effect of systematic weight training on athletic power. Res Quart, 21: 188-194, 1950.

143. Clarke DH: Correlation between the strength/mass ratio and speed of an arm movement. Res Quart, 31: 570-574, 1960.

144. Clarke DH: Adaptations in strength and muscular endurance resulting from exercise. Exercise Sport Sci Rev, 1: 73-102, 1973.

145. Clarke DH, Herman L: Objective determination of resistance load for ten repetitions maximum for quadriceps development. Res Quart, 26: 385-390, 1955.

146. Dons B, Bollerup K, Bonde-Paterson F, Hanckes: The effect of weight-lifting exercise related to muscular fiber composition and muscle cross-sectional area in humans. Eur J Appl Physiol, 40: 95-106, 1979.

147. Noble L, McGraw LW: Comparative effects of isometric and isotonic training programs on relative load, endurance and work capacity. Res Quart, 44: 96-108, 1973.

148. Stone JH, Johnson RL, Carter DR: A short term comparison of two different methods of resistance training on leg strength and power. Athl Training, 14: 158-160, 1979.

149. Hakkinen K, Alen M, Komi PV: Neuromuscular, anaerobic, and aerobic performance characteristics of elite power athletes. Eur J Appl Physiol, 53: 97-105, 1984.

150. Lesmes GR, Fox EL, Stevens C, Otto R: Metabolic response of females to high intensity interval training of different frequencies. Med Sci Sports, 10: 229-232, 1978.

151. Fox, EL, Bartels RL, Klinzing J, Ragg: Metabolic responses to interval training programs of high and low power output. Med Sci Sports, 9: 191-196, 1977.

152. Hunter GR, McCarthy JP: Pressor Response associated with high-intensity anaerobic training. Phy Sport Med. 11: 151-162, 1983.

153. Houston ME, Thomson JA: The response of endurance - adapted adults to intense anaerobic training. Eur J Appl Physiol, 36: 207-213, 1977.

154. Cunningham DA, Faulkner JA: The effect of training on aerobic and anaerobic metabolism during a short exhaustive run. Med Sci Sports, 1: 65-69, 1969.

155. Thorstensson A, Sjodin B, Karlsson: Enzyme activities and Muscle strength after "sprint training" in Man. Acta Physiol Scand, 94: 313-318, 1975.

156. Roberts AD, Billeter R, Howald H: Anaerobic muscle enzyme changes after interval training. Int J Sports Med, 3: 18-21, 1982.

157. Weltman A, Moffatt RJ, Stamford BA: Supramaximal training in females: Effects on anaerobic power outputm anaerobic capacity, and aerobic power. J Sports Med, 18: 237-244, 1978.

158. Hempel LS, Wells CL: Cardiorespiratory Cost of the Nautilus Express Circuit. Phy Sports Med, 13: 82-96, 1985.

159. Hurley B, Seals D, Hagborg J, et al: High-Density-Lipoprotein Cholesterol in Body builders v Powerlifters. J Am Med Assoc, 252: 507-513, 1984.

160. Goldburg L, Elliot D, Schutz R, Kloster F: Changes in Lipid and Liporotein Levels After Weight Training. J Am Med Assoc, 252: 504-506, 1984.

161. Lamb D: Physiology of exercise, Responses and adaptations. Macmillan Publishing CO, New York, 1978.

162. O'Shea JP: Science principles and methods of strength fitness Addison-Wesly Publishing Co, Reading MA

163. Westcott WL: Strength fitness, phsiological principles and trainin technigues. Allyn and Bacon Inc.,Boston, 1983.

164. Strauss RH, Liggett MT, Lanese RR: Anabolic steroid use an preceived effects in ten weight-trained women athletes. J Am Me Assoc, 253: 2871- 2873,1985

165. Green HJ, Houston ME, Thomson JA, Fraser IG: Fiber Type distributic and maximal activities of enzymes involved in energy metabolis following short-term supramaximal exercise. Int J Sports Med. 5: 198-201, 1984.

166. Wilmore JH: Training for sport and activity the physiologica bases of the conditioning process 2 nd ed. Allyn and Bacon Inc. Boston, 1982.

167. Fahey T, Rolph R, Moungmee R, Nagel J, Mortana S: Serum testosteron body composition and strength of young adults. Med Sci Sports 8:31-34, 1976.

168. Hurly B, Seals D, Ehsani A, et al: Effects of high-intensity strength training on cardiovascular fuction. Med Sci Sport: Exercise, 10:13-15, 1978.

169. Lukaszewski J, Biczowa B, Bobilewicz D, Wilk M, Obuchowics-Fiedlus: Effect of physical exercise on plasma cortisol and growth hormon levels in young weight lifters. Enokrynologia Polska XXVII, 2: 149-158, 1976.

170. Shephard R, Sidney K: Effects of physical exercise on plasma growth hormone and cortisol levels in human subjects. Exercise Sports Sci Rev, Academic Press Inc., New York, 1975.

171. Skierska J, Ustupska J, Biczoa B, Lukaszewski J: Effect of physica exercise on plasma cortisol, testosterone, and growth hormone level in weight lifers. Endokrynologia Polska XXVII, 27: 159-165, 1976.

172. Terjung R: Endocrine response to exercise. Exercise Sport Sci Rev, 5:1979.

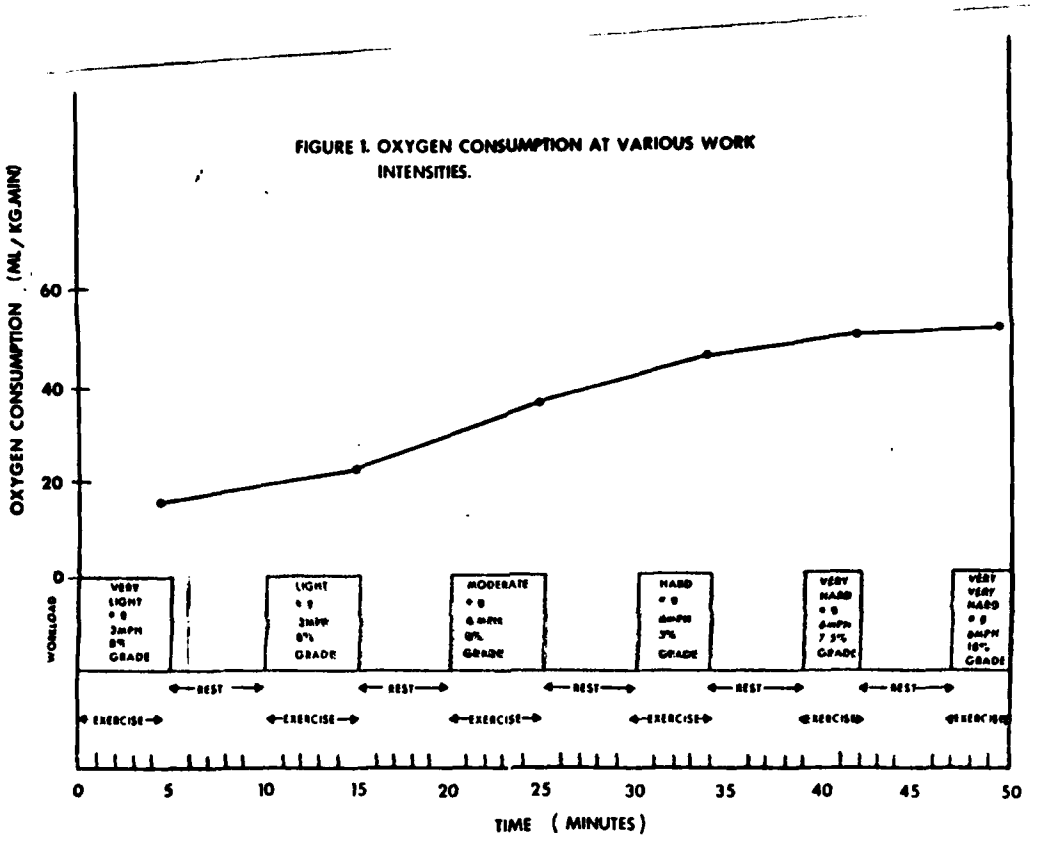
173. Vahelder W, Radomsk M, Goode R: Growth hormone response during intermittent weight lifting exercise in men. Eur J Appl Physiol. 53: 31-34, 1984.

174. Saltin B, Nazar D, Costill L, et al: The nature or the training respose; peripheral and central adaptions to one legged exercise. Acta Physiol Scand, 96: 298-305, 1976.

175. Weiss L, Cureton J, Thompson F: Comparison serum testosterone and rostenodione responses to weight lifting in men and women. Eur Appl Physiol, 50: 413-419, 1983.

176. MacDougall JD, Sale, DG, Alway SE, and Sutton JR: Muscle fiber number in biceps brachii in bodybuilders and control subjects. J Appl Physiol: Respira Environ Exercise Physiol 57:1399-1403,1984

FIGURE 1. OXYGEN CONSUMPTION AT VARIOUS WORK INTENSITIES.



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